An Attempt to Prevent Winter Depression by Light Exposure at the End of September

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An Attempt to Prevent Winter Depression by Light Exposure at the End of September

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Key Words: Seasonal affective disorder, prevention, light treatment

Introduction

Seasonal affective disorder (SAD), winter type, is characterized by the occurrence of depression in autumn/winter, followed by a complete recovery in spring/summer. In SAD patients, some atypical symptoms are highly characteristic, such as hypersomnia, carbohydrate craving, weight gain, fatigue, and loss of social interactions (Rosenthal et al 1984). The repeated occurrence of symptoms during successive winters is one of the diagnostic criteria. Not every SAD patient becomes depressed every year, however. In prospective studies it was found that 70.4% of the SAD diagnosed subjects who were followed from a symptom-free moment at the end of September, became depressed during the following winter (Meesters et al 1991; 1993a).

In a previous study it was found that severe winter depression can be prevented by light treatment administered at the first signs of an emerging winter depression, that is, the moment subjects reported complaints of slight depression (Meesters et al 1991; 1993a). In the present study, the question was raised whether light exposure given at the beginning of the winter season, when subjects are still free of symptoms, could be successful in preventing the development of a winter depression during the rest of the season. Such a treatment, which might prevent depression altogether, would be highly advantageous.

Methods and Results

Fifteen SAD patients, all diagnosed according to the criteria of Rosenthal et al (1984) gave their consent to participate in the study. They had been drug free for at least 1 month prior to the study, were without physical complaints, and had reacted with a full remission to light treatment in the previous winter season. They received light exposure in the clinic (2500 lux, Philips TL 58 w/95). The end of daylight saving time (Central European Time, September 29, 1991) fell within the time schedule of the study. Light exposure was administered at the same time in the natural light/dark cycle for all patients, that is, for five patients from 10.00 AM till 1.00 PM before the change of the clocktime and for six patients from 9.00–12.00 AM after the time change. In this way the timing of light treatment equalled that used in our previous 'prevention' study (Meesters et al 1991; 1993a). The course of mood was assessed every week during the entire winter season from the September 7, 1991 through of April 15, 1992 by means of the Beck Depression Inventory (BDI, 21-item version) (Beck at al 1961, 1979; Bouman et al 1985). In analogy to the addendum to the Hamilton Rating Scale for Depression (Rosenthal and Heffernan 1986), we created an addendum to the BDI (BDI-ADD), in order to assess atypical symptoms by means of self-ratings (Meesters and Jansen 1993b). This addendum contains the items 12 (social withdrawal) and 17 (fatigue) from the BDI, and the inversely formulated items 16 (hypersomnia), 18 (appetite) and 19 (weight gain).

There were four drop-outs: two subjects showed signs of depression before light treatment was started and two other subjects did not complete the design because of the unpleasant obligation of having to perform weekly mood ratings. Therefore, data from 11 subjects were available for analysis, 1 man, age 36, and 10 women, mean age 37.7, SD ± 9.9. As an inclusion criterion we used a score on the BDI <13, during the 2 weeks before light exposure. After light exposure, subjects completed the BDI at home and sent it to the clinic. If they reached a BDI ≥22 at a particular moment, or a BDI score ≥13 for 2 consecutive weeks, they were considered to be depressed and were therefore offered light treatment.

Eight out of 11 subjects (= 73%) reached this criterion. Figure 1 shows in which month the subjects became depressed. The time

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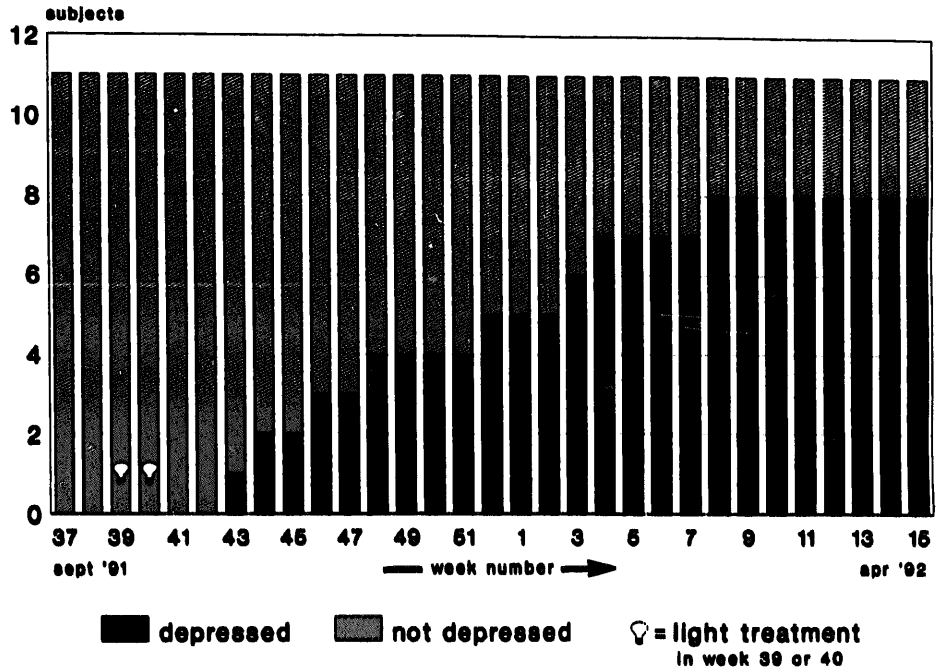

Figure 1. Cumulative number of subjects who became depressed (2 consecutive scores on the Beck Depression Inventory (BDI) ≥ 13, or a score of BDI ≥ 22) during the season after an initial light treatment at the end of September. At that time subjects were free of symptoms.

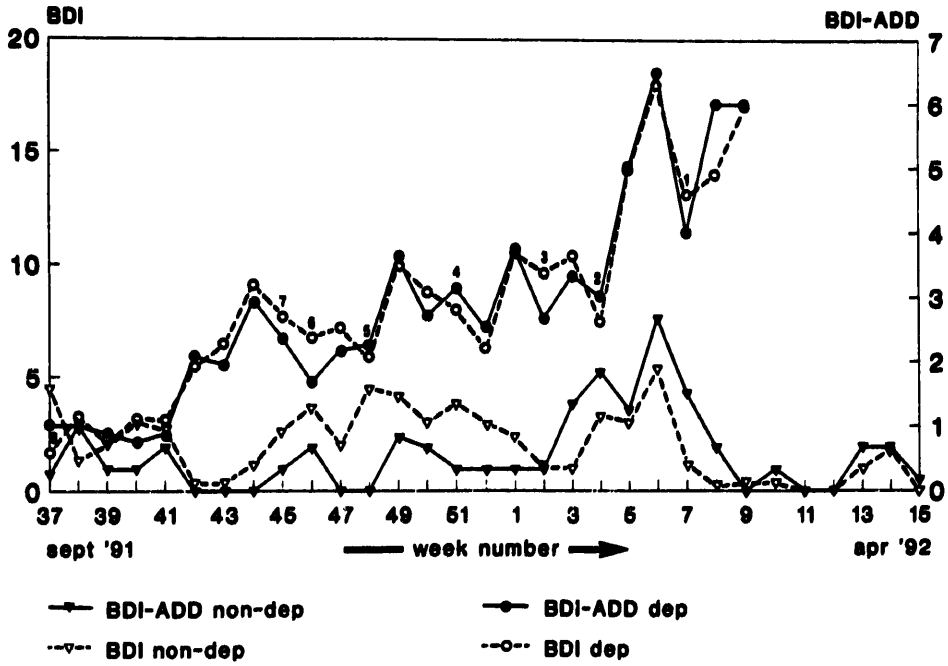

Figure 2. The course of mood (Beck Depression Inventory, BDI) and the course of atypical symptoms (Beck Depression Inventory—Addendum, BDI ADD) during the winter season. Non-dep: subjects who did not become depressed ($n = 3$); Dep: subjects who became depressed. Numbers of individuals contributing to the curve are indicated for the depressed group. The reason for the decreasing number of subjects as time progressed in the depressed group is that subjects were removed after they became depressed, and therefore got light treatment.

interval between the initial treatment, at the end of September, and the development of depression varied from 2 to 19 weeks (mean 9.6 weeks). The course of depression and the course of atypical complaints are shown in Figure 2, both for the group of subjects that reached the criterion for depression and for the group that did not. Obviously, light exposure early in the winter season did not prevent the occurrence of depressive episodes. The time course of depression is very similar to the time course of atypical complaints in both groups.

For all 11 subjects, significant correlations ($p = <0.05$) between individual BDI and individual BDI-ADD scores were found (range Pearson r: 0.464–0.983; range n: 8–31). After removing the items 12, 16, 17, 18, and 19 from the BDI, because of their dependency with the BDI-ADD scores, the correlations between the remaining part of the individual BDI and the individual BDI-ADD scores are still significant ($p = <0.05$; range Pearson r: 0.509–0.976). using BDI-ADD scores ≥ 4 for 2 consecutive weeks as a criterion for the incidence of a depressed period, there was 100% correspondence between the criteria based on the BDI and those based on the BDI-ADD.

Discussion

An attempt to prevent the development of winter depression by light exposure at a symptom-free period at the beginning of autumn was not successful. The percentage of subjects who became depressed (73%) was comparable to that found by Thompson (1989), who reported that 67% of the SAD subjects diagnosed in the summer became depressed during the following winter and by Meesters et al (1993a) who reported that 70.4% of SAD-diagnosed patients became depressed when they were followed from a symptom-free period (summer). It is hard to tell whether this light treatment had any delaying effects on the course of depression, as no control group was studied. Two subjects reported spontaneously that the beginning of their depression was substantially later in the season, as compared to their experiences from the previous winters. Moreover, the possibility cannot be ignored that exposure to more light (longer, higher intensity) or to light exposure at other moments of the day may have had the effect of preventing a depressive episode.

The present results differ substantially from the results of a previous study (Meesters et al 1991, 1993a). There it was found that the development of a full-blown winter depression could be prevented by light treatment applied at the first signs of depression. In contrast to the present study, no severely depressive episode was observed during the remaining part of the season. If the effects of this study can be replicated in future studies, these discrepancies may perhaps be explained from state-dependent sensitive to light treatment; at the first seasonal occurrence of symptoms patients could still be very sensitive to light treatment, whereas a complete recovery may be more difficult to achieve when a depression has persisted for a longer period of time. This hypothesis might also explain why studies vary considerably in the rate of relapse. Whether this is related to the phase in the depression episode at which light treatment is applied, needs further investigation.

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